

# NEUROPSYCHOLOGY TODAY

*Monthly Newsletter published by Dr. Danov Neuropsychologist, P. C.  
November 2009 Issue – Adult Neuropsychology: Alcoholic Encephalopathy*

## Alcohol Use in the US

Alcohol abuse is among the most significant health problems in the world, as it has been linked to such conditions as cancer, and liver and heart disease. It is estimated that 40 million people are affected by alcoholism in the US, and 20-40% of all inpatients are hospitalized due to illnesses caused or exacerbated by alcohol. Patterns of drinking world-wide are changing, as increasingly more women and young people drink heavily.<sup>1</sup>

Individuals who abuse alcohol for substantial period of time may develop dependence. Withdrawal symptoms occur within 4-12 hours of abstinence and range from anxiety, irritability, increased heart rate, perspiration, and insomnia to seizures and hallucinations. Some individuals experience acute cognitive symptoms after cessation of alcohol use, which may or may not resolve with time.<sup>1,2</sup>

Alcoholism can lead to such conditions as alcoholic encephalopathy and Korsakoff's syndrome, alcohol-induced persisting dementia, and hepatic encephalopathy. These illnesses have been linked to significant cognitive impairment, the extent of which can be accurately assessed by neuropsychological exam.<sup>1,2</sup>

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## Alcoholic Encephalopathy: Neurophysiology

Alcoholic Encephalopathy (AE), also referred to as Wernicke's Encephalopathy is a medical condition that results from a deficiency in thiamine (vitamin B1), which is most frequently caused by excessive alcohol use. More males are diagnosed with this neuropsychiatric disorder compared to females.<sup>3,4</sup>

Healthy level of thiamine is essential for normal brain functioning, since this coenzyme mediates such neurochemical brain processes as carbohydrate and lipid metabolism and aminoacid production. Low levels of thiamine lead to an impairment in cellular energy metabolism, resulting in damage in selective diencephalic and brainstem areas. It has been documented that hemorrhagic lesions can develop in

*(Continued on Page 2)*



## Symptoms and Diagnosis of Alcoholic Encephalopathy

AE is typically characterized by an acute onset of a "classic triad" of symptoms, including:

1) psychological and cognitive changes (apathy, agitation, hallucinations, behavioral disturbances, confusion, slowed thinking, poor concentration, and impaired awareness of the immediate situation)

2) ocular abnormalities

3) motor problems (gait incoordination and ataxia).<sup>3,4</sup>

AE is greatly under-diagnosed. It is reported that only 20-25% of autopsy cases found to have AE have been diagnosed with this condition in their lifetime. This can be attributed to a lack of a specific test that can point to AE. Additionally, the diagnosis among the alcoholics is complicated by the fact that the symptoms of WE are virtually impossible to differentiate from drunkenness.<sup>3</sup>

MRI is currently among the primary methods to confirm AE diagnosis (see an MRI image of marked structural brain changes on p.3). Some measures that can also be helpful in diagnosing AE include testing blood for thiamine level and red blood cell transketolase activity. A relatively new liquid chromatography method for the assessment of thiamine has been found promising in helping diagnose individuals with AE.<sup>3</sup>

*("Wernicke's Encephalopathy: Neurophysiology," continued from p. 1)*

as little as 2-3 weeks of thiamine deficiency.<sup>3,4</sup>

In addition, it has been established that individuals with AE have reduced brain weight due to brain atrophy, which is mostly accounted by a reduction in the white matter. The connection between alcohol and brain damage is further supported by a correlation between the amount of alcohol consumed over a lifetime and the degree of brain atrophy.<sup>1</sup>

Further, AE is frequently associated with beriberi, a condition marked by severe lethargy and fatigue, together with cardiovascular, nervous, muscular, and gastrointestinal complications. Some people with AE develop cardiovascular (wet) beriberi, which can cause heart failure and edemas. Others are found to have Central Nervous System (dry) beriberi, described as paralysis resulting from damaged peripheral nerves.<sup>4</sup>

Since alcohol affects the breakdown of thiamine in the body, AE diagnosis is typically associated with alcoholism. Other conditions that have been found to cause Wernicke's encephalopathy include gastrointestinal disorders, certain cancers, thyroid and renal diseases, and AIDS. AE is less likely if an alcoholic person maintains a healthy and nutritious diet. If untreated, AE can lead to coma and death.<sup>3,4</sup>

### **Alcohol-Induced Conditions**

A number of illnesses caused by excessive alcohol use can result in cognitive decline. Using objective test measures, neuropsychologist can differentiate between various types of cognitive impairment such as dementia, pure amnesia, and mild cognitive impairment,

give an appropriate diagnosis and treatment recommendations, and monitor the progression of cognitive symptoms over time.

### **Korsakoff's Syndrome**

About 80% of individuals with AE develop Korsakoff's syndrome, which is characterized by a severe impairment in short-term memory and anterograde amnesia. Although typical Korsakoff's syndrome is described as a pure amnesic state without significant intellectual decline, long-term memory loss, executive functioning deficits, disorientation to time, and overall dementia can develop.<sup>2,5</sup>

Additionally, patients with Korsakoff's syndrome tend to make up stories (confabulation) rather than admitting memory loss. They may lack insight with regard to their memory impairment, and are sometimes completely unaware of it. Korsakoff's patients have also been described as having little to say in spontaneous conversations, as well as presenting with apathy, indifference, and incapacity to persevere in ongoing activities.<sup>2,5</sup>

Timely treatment of AE with thiamine can prevent the development of Korsakoff's syndrome. However, once developed, Korsakoff's syndrome is non-responsive to thiamine therapy. Because AE is typically followed by Korsakoff's syndrome, and both appear to share common neuropathology, they are frequently viewed as a continuum described as Wernicke-Korsakoff syndrome.<sup>2,5</sup>

### **Alcohol-Induced Persisting Dementia**

It is estimated that about 25% of elderly alcoholic patients and 24% of all institutionalized elderly suffer from alcohol-induced dementia. This condition is characterized by dementia that persists long after alcohol consumption has ceased.

The symptoms include impaired general intellectual ability, memory, visuospatial, abstract reasoning, and problem-solving abilities, while language is generally preserved.<sup>2</sup>

### **Hepatic Encephalopathy**

As many as 30% of people who abuse alcohol develop cirrhosis of the liver. The resulting impaired protein synthesis can cause psychomotor, language, perceptual impairments, while decreased hepatic blood flow can lead to decreased language efficiency. Many cognitive deficits improve after liver transplantation, although memory impairment does not.<sup>2</sup>

#### **About Dr. Rimma Danov**

Dr. Rimma Danov received her PhD in clinical psychology from Adelphi University in NY. She completed her internship in clinical psychology and neuropsychology at Harvard Medical School and postdoctoral fellowship in pediatric and adult neuropsychology in a private clinic affiliated with NJ Medical School and the Robert Wood Johnson Medical Center. She is an assistant clinical professor at Penn State University, Dept. of Kinesiology, and has served as an assistant clinical professor at NYU School of Medicine, Dept. of Neurology, and Adelphi University, Derner Institute. In the past, she worked as a neuropsychologist for the NJ Devils Hockey Team and was engaged as a co-investigator of TBI in boxers at the NYS Athletic Commission.

Presently, Dr. Danov maintains a full-time private neuropsychology practice where she examines neurocognitive and neurobehavioral functioning of patients 2-90 years of age with various neurological and neuropsychiatric disorders, such as MS, TBI, CVA, Parkinson's, Alzheimer's, dementia, ADHD, PDD, Autism, learning disabilities, seizures, and many others, using state-of-the-art neuropsychological techniques. Dr. Danov also conducts and publishes research in these areas. She is available for medico-legal consultations and testimony.

## College Drinking and Cognitive Functioning

Excessive drinking affects virtually all college campuses, their students, and the surrounding communities.<sup>1</sup> According to the American Medical Association, almost 20% of 12-20 year-olds report binge drinking, defined as having 4-5 drinks in a row.<sup>6</sup>

Previously, the common view held that the young brain is more resilient to the effects of alcohol. However, we now know that alcohol takes a greater toll on brain development of individuals under the age of 21 than on any other age group. Moreover, adults would have to consume twice as many drinks to suffer comparable damage as adolescents, and even occasional heavy drinking injures young brains.<sup>6</sup>

During adolescence, the brain goes through dynamic changes, such as frontal lobe development and the refinement of pathways and connections. Alcohol can disrupt these processes and cause long-term and irreversible damage to the brain. Even short-term or moderate drinking during adolescence can impair learning and memory.<sup>6</sup>

With regard to neuropsychological findings, research shows that adolescent drinkers perform worse on vocabulary, general information, visuospatial, and memory tests compared to their non-drinking peers. The most dramatic differences are seen in verbal and non-verbal recall.<sup>6</sup>

Neuropsychological exam can objectively assess all cognitive functions to determine

which functions have been negatively affected by excessive drinking. Such neuropsychological profile is used to understand how students' learning skills have been weakened by drinking. It is also used to develop cognitive remediation treatment plan and to track improvement of these cognitive skills with treatment.

Adolescents who drink heavily also get lower grades in school and are more likely to experience social problems, depressive symptoms, suicidal thoughts, and violence. Their sleep cycle may be affected, resulting in impaired learning and memory, as well as disrupted release of hormones that play an important role in teenagers' growth and maturation. Alcohol can also increase the risk of stroke among young individuals.<sup>6</sup>

Based on the neuroimaging and neuropathology data, teenage drinkers tend to have a reduced volume of hippocampus, an area linked to memory and learning. Additionally, the prefrontal area of their brain, which is important in the formation of adult personality and behavior (consequential thinking, reasoning, impulse control, effortful attention, etc.) may

be marked by significant structural changes.<sup>6</sup>

Even though some mild and moderate adolescent drinkers may be able to catch up in adulthood, heavy drinkers may never be able to reach the level of cognitive development of their non-drinking peers. Rather than "outgrowing" excessive alcohol use, young drinkers are much more likely to have drinking problems as adults. Ultimately, alcohol may impede these individuals' ability to achieve the goals of young adulthood such as marriage, educational attainment, employment, and financial independence.<sup>6</sup>

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1. Background image (pp.1,4): Jeff Johnson Biolog. & Medic. Visuals
2. Alcoholic (p.1): alcoholrehab247.com
3. MRI (p.3): From Works cited #4 above.

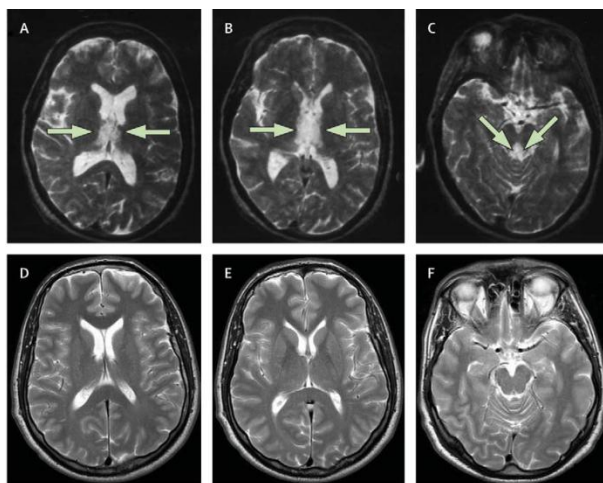
### Editor

Dr. Rimma Danov, Ph.D.

### Layout:

Natalia Shtompel, M. A.  
Research Coordinator

**Next Issue-** Dec'09: Premature Children; Jan'10: Mild Cognitive Impairment (MCI)



T2-weighted axial MRI above shows lesions in the medial thalami (A,B) and in the grey matter of the midbrain (C) in a patient with Wernicke's encephalopathy 2 weeks after onset of neurological symptoms vs. a healthy brain (D,E,F).

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**Languages**

We are very much open to diverse cultures in this practice and value the quality of a bilingual neuropsychological exam performed in the patient's native language. Dr. Danov is a native Russian speaker. Her current clinical staff include native **Russian, Spanish** and **Hebrew** speakers.

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**Dr. DANOV NEUROPSYCHOLOGIST, P.C.**

65 Kelvin Avenue  
Staten Island, NY 10306

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